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EXPLORING TROPICAL RICE DISEASES: A REMINISCENCE

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INTRODUCTION

I never expected to have the privilege and honor of writing a prefatory chapter for the *Annual Review of Phytopathology*. I appreciate the editorial committee's broad perspective, which extends over the people and problems of plant pathology in nations all around the world, and I treasure this opportunity.

Much has been written by eminent scientists on the philosophy, concepts, principles, and directions of plant pathology, as well as on the attitudes of plant pathologists; I have little to add. It is easier to say something of one's past. More than half of my 50 years as a plant pathologist was the turbulent period of civil wars and World War II. During the early years research programs were disrupted and often changed. As a result, the most peaceful, interesting, and rewarding time in my career has been my years of association with the International Rice Research Institute (IRRI) in the Philippines. I want to recount some of the events that I believe are significant in the development of rice pathology in the tropics, the details of which are largely unknown to most people. Hindsight is often helpful; L. M. Black said: "Learning is much more likely to be derived from history than from prophecy" (1).

The recent history of rice pathology in the tropics reflects the general state of plant pathology in many tropical nations. The number of plant pathologists has been insufficient to cope with important disease problems. Mistakes have been made through prejudice, misconception, and blind faith in old ideas and untested new ones. Rice is an important crop to the hungry people of the world, yet it was long neglected by the world scientific community.

Rice is a remarkable plant. It has great antiquity and a very broad and diverse genetic base in its many thousands of cultivars and landraces. The genetic

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diversity of rice provides numerous genes for resisting many pathogens. We are convinced that most rice disease problems in the tropics can be resolved with genetic resistance. In its long history of cultivation in broad regions, rice has been associated with all types of pathogens. Many deter rice production and until recently were very little known. Here are the results of our explorations into some of these problems.

MENTEK AND PENYAKIT MERAH

A rice malady called mentek was known in Indonesia as early as 1859, and severe losses were reported between the 1930s and 1950s. For many years Dutch scientists studied the disease intensively from the soil chemistry and nutritional point of view, working on the hypothesis that it was a physiological disorder. However, to their credit, in spite of detailed studies they never concluded that it is physiological. Later they studied nematodes as a possible cause of the disease.

Penyakit merah is another disease known in Malaysia since 1938. Several foreign experts were invited to study it for several years. Perhaps influenced by the study in Indonesia, it was also considered physiological and was reported to be due to a metabolic deficiency of nitrogen. Similar but less known is so-called suffocation disease, seen in Taiwan in the early 1960s, said to be due to a lack of oxygen in the soil. All of these diseases turned out to be due to virus infections. Mentek and penyakit merah were later known as tungro disease; suffocation disease was called transitory yellowing.

Many plant pathologists know about cadang-cadang disease of coconut in the Philippines, but very few know there is also rice cadang-cadang disease. Rice cadang-cadang disease was first reported in 1939 and caused some 30% loss of rice nation-wide in the 1940s. Researchers suspected that it had a viral nature and thought it similar to Japanese dwarf disease. It was later shown to be due to the tungro virus.

Even though virus diseases of rice had occurred in the tropics for a long time in different places, almost no reliable information on them was available when the IRRI began to operate. When the first large batch of several thousand rice germ plasm collections was planted at an IRRI farm for seed multiplication in 1962, there was a great display of reddish, orange, yellow, and dark green leaf discoloration. Many of the entries were stunted. Because the physiological disease hypothesis was still strong, some thought that these were physiological disorders. However, extensive field observations showed that the discolorations occurred in upland rice as well as in the lowlands. Sometimes among seedlings in the same hills some were healthy while others showed symptoms. Thus, the disorder was not likely to be physiological. We therefore turned our attention to its possible viral nature and during the next two years identified

three new virus diseases, which we called orange leaf, tungro, and grassy stunt (4).

Even after positive identification of the viruses, some continued to think of the diseases as physiological. One day the leader of a rice team from an international organization in Manila invited us to see an experiment developed by their plant physiologist, who claimed that it proved that tungro in his potted plants was due to a nutritional disorder. This was an interesting challenge to our report. Several plant physiologists and pathologists, as well as reporters and photographers, gathered in the developer's laboratory. What we found in his pots, however, were seedlings showing simple nitrogen deficiency symptoms, not the tungro disease of the field. After adding a little nitrogen fertilizer, the plants returned to normal. That plant physiologist left Manila soon afterward.

In 1964 I visited Malaysia and had an opportunity to see penyakit merah in the field firsthand. It struck me as being very much like tungro disease. We dispatched one of our staff to live in a small agricultural experiment station, taking with him a few small insect cages and equipment for transmission studies. In less than two months, he obtained proof that penyakit merah was indeed the same as tungro. The symptoms, the vector, and the manner of transmission were the same. The experiments did not convince everyone, however, and for many years some pursued evidence that penyakit merah is a physiological disorder.

In 1965, we set up a simple field test that served as a demonstration plot as well. In a diseased field found early in the season, a dozen microplots of 2×2m were set up side by side. The diseased young plants were removed and replaced with new seedlings, some of which were healthy (raised in a greenhouse) and some of which were artificially inoculated with tungro virus. Some of the plots were covered with insect cages and some were exposed. At the end of the season, the results were dramatic. Plots with healthy seedlings covered by the insect cages grew normally and gave normal yields. Those inoculated or those in the exposed plots showed typical symptoms of penyakit merah (tungro) and yielded only a few hundred kilograms per hectare. This changed the attitudes of many people both in and outside Malaysia.

The identification of penyakit merah of Malaysia as a viral disorder led me to suspect that mentek of Indonesia was also a virus disease similar to tungro. This proved to be true in 1967, when we were able to travel to Indonesia. Transmission experiments were again conducted and positive evidence was obtained.

In 1965, one of our students found tungro when he returned to Thailand, and in 1966–1967 it affected one-third of the rice in that country. India began to survey for virus diseases in 1967 and eventually found them to be widespread in many states. It became apparent then that virus diseases were major pathological problems of rice in the tropics.

Many other virus and virus-like (mycoplasma) diseases of rice have been

reported since 1970; e.g. necrosis mosaic (Japan), yellow mottle (Kenya), giallume (Italy), waika (Japan), wilted stunt (Taiwan), ragged stunt (Indonesia, Philippines, Thailand, India), bunchy stunt (Mainland China), gall dwarf (Thailand), chlorotic streak (India), and crinkle (West Africa). More will probably be found in the future. The discovery of these new virus diseases and the fact that they appeared in different locations led me to think that the viruses might come from wild graminaceous hosts.

In the early 1950s, Dutch rice breeder J. G. J. van der Meulen developed several cultivars (e.g. Tjeremas, Bengawan, Peta, Intan, Sigadis) from a well-known cross between Latisail and Tjina in Indonesia. These cultivars were resistant to mentek, even though the nature of mentek was not known at the time.

In rearing the green leafhopper for tungro transmission studies at IRRI, K. C. Ling hypothesized that the cultivar Peta would be a better host for feeding and ovipositioning the insects because its leafsheath is longer and larger than that of the conventionally used Taichung Native 1 (TN1). However, the results were the contrary. Insect populations on Peta plants were much smaller than those on TN1. This was the first recognition of leafhopper resistance in rice, now an accepted fact of great importance in rice insect control. Many other discoveries were incidental. The diverse genes with which rice resists both viral pathogens and vector insects should enable us to protect new cultivars from heavy damage.

BACTERIAL LEAF STRIPE AND KRESEK

In the early days bacterial diseases of rice in the tropics were considered unimportant and reports on them were very scarce. O. A. Reiking in 1918 reported a bacterial leaf stripe disease from the Philippines that produced narrow stripes with a watery, dark green, translucent appearance. He did not identify the causal organism. Many authors have erroneously referred to the disease Reiking described as bacterial leaf blight. It was not until 1957 that this bacterial leaf streak disease, as it is now called, was differentiated from bacterial blight during a study in Mainland China.

In 1950 J. Reitsma & P. S. H. Schure described a bacterial disease of rice in Indonesia called kresek, and in 1953 Schure identified the organism that causes it, *Xanthomonas kresek*. *Kresek* is a local word, we were told, that describes the dead rice plant with its leaves floating on the water in the field. The symptoms were very different from those of bacterial leaf blight known in Japan.

In 1964, Masio Goto came to the IRRI as a visiting scientist. In this capacity, he visited many countries in Southeast Asia, including Indonesia, where he had an opportunity to closely scrutinize kresek disease. He noticed typical bacterial leaf blight symptoms mingled with kresek symptoms and suspected that the two

might be the same disease. He was excited by this observation and the evening he returned from Indonesia said to me that we might find kresek right in the IRRI plots. We found it the next morning. The symptoms that all along we thought were due to injuries from stem borer, so-called dead heart, were actually symptoms of kresek. Later he proved experimentally that leaf blight bacterium caused the kresek symptoms. Since his report, kresek has been found in many countries, not only in the tropics but also in Korea and Japan. Presumptions, prejudices, and belief in the pronouncements of others are so often impediments to finding the truth!

In addition to kresek, Goto described another distinct symptom of bacterial blight in the tropics, the pale yellow syndrome. One or two young expanded leaves of normal size on some of our field-grown plants had partly or entirely turned a pale yellow color while the other leaves stayed green. A few of the lowest leaves often died. We had had the pale yellow plants brought to our laboratory before Goto described the symptoms. There was one post-doctoral visiting scholar who wanted to see if the disease was viral in nature. Since he had experience with tobacco mosaic, he comminuted the leaves and rubbed the juice on leaves of healthy seedlings, just as he did when inoculating tobacco with tobacco mosaic virus. A few weeks later he showed me the inoculated plants, saying he thought his inoculation experiment had succeeded in transmitting a virus. I was not convinced. After three repeated trials, he got the idea that I was preventing him from publishing a new discovery. Soon afterward, Goto produced results with bacterial inoculation. Goto's reports opened up new horizons in tropical bacterial disease research. Cultivar IR8 was becoming popular in the late 1960s, and its susceptibility to bacterial blight aroused further interest in the disease.

For a long time bacterial blight was known as an Asian disease. Only recently has it been reported in Africa, Latin America, and, a little earlier, from Australia. The origin of the disease in areas outside Asia is of interest. Seeds have been reported as the method of transmission in temperate regions, but we failed to find seed transmission in the hot and humid tropics. In the tropics the bacterium dies quickly, and even in an active leaf lesion living bacterial cells seem to exist only in the peripheral areas. Isolations cannot be made from the center or older portions of the lesion. Seeds of rice cultivar IR8 and others have been sent to Africa and Latin America for several years, sometimes in large quantities, but the leaf blight disease has never been reported. In 1976 I visited Central America and found a few rice plants showing symptoms of bacterial blight in an isolated, newly developed rice area. I also observed many typical bacterial leaf blight-like symptoms on at least four species of wild graminae in large areas nearby. I speculated that the bacterium on rice may come from the wild graminae. The presence of the disease on rice was confirmed later by Lazano in Latin America (4), but he believed that the disease was probably

introduced through imported seeds. Yet relatively few rice seeds are imported into Australia and the strictest quarantine probably prohibits introduction of the disease. For several years I. W. Buddenhagen did not observe the disease in Africa but reported its presence recently. He also suspects that it is of local origin.

PHYSIOLOGIC RACES OF *PYRICULARIA ORYZAE*

The presence of pathogenic races of rice blast fungus, *Pyricularia oryzae,* was noticed as early as 1922 in Japan; the reactions of cultivars to the fungus differed on the plain and in the hilly areas. The phenomenon was demonstrated easily by inoculation experiments. During the 1950s and 1960s, Japanese and US workers studied physiologic races intensively, and many other countries began to identify their races in the 1960s. Imitating the procedures that began with cereal rusts, a set of 10–12 differential cultivars were inoculated with pure culture isolates of the fungus, producing a table of resistance and susceptibility grouped into races. Several investigators have spent 15 or more years on the subject. A great deal of effort has been expended trying to find out what races exist in this or that country.

To establish a rice cultivar as resistant (R) or susceptible (S) to an isolate of the blast fungus is not always easy. During earlier studies, some investigators complained that certain differential cultivars were not good because they produced different types of lesions, from resistant to susceptible on a single leaf. Others found some fungus isolates inconsistent, producing one pattern of reaction (race) on test cultivars in the first inoculation but another pattern in the next inoculation.

We started the race study as everyone else did. We also found the different lesion types on the same leaves and very different lesion numbers among the cultivars. In our inoculation experiments, among 20 seedlings of each differential cultivar there may have been numerous lesions on each seedling of some cultivars, while other cultivars showed only a few lesions on a few of the seedlings. In these cases, the designation of cultivar as resistant or susceptible depended on the discretion of the investigator. In many instances, a leaf may have had few large S-type lesions but many more R-type lesions. If typical S-type lesions were present the cultivar would be conventionally considered susceptible, but this did not seem to be logical. The expression of reaction was often quantitative. Simple R and S signs to indicate the reactions between the differential cultivars and the isolates seemed grossly incomplete and arbitrary.

We began our race study by employing the Japanese and the US as well as the Taiwanese differential sets. After a hundred inoculations using the US set, we identified some 20 races in the conventional manner. Based on the US set we found a race, say race X, consisting of 17 isolates. To six of them a local popular cultivar, Peta, was susceptible and to the other 11 isolates Peta was

resistant. If someone asked whether Peta was susceptible or resistant to that race X, what would be our answer? The existing sets of differentials were not useful in the Philippines.

To search for a new set of differential cultivars, we started with a broad base, selecting 110 rice cultivars, including the Japanese, Taiwan, and US differentials as preliminary candidates. After inoculating these 110 cultivars with 50 isolates, two things among others were apparent: (*a*) some cultivars were susceptible to none or only a few of the isolates, while others were susceptible to as many as 90% or more of the isolates, and all gradations from zero to 100% were found, (*b*) no two isolates had the same reactions on all the cultivars; they were all different.

It became clear that conventional race grouping is an oversimplification that had led to a common misconception among rice workers that the isolates belonging to a race are identical. Often investigators have selected one of many isolates as representative of the race for testing the resistance of new cultivars. In fact, it may not be representative, as shown in the case of the isolates of race X and Peta above. The results also showed that the number of races identified depends not only on the number of differential cultivars, but also depends on what cultivars are used, as we all know. If one selects the cultivars with the highest resistance or susceptibility reactions, there will be few races. If one selects those with both resistance and susceptibility in approximately equal numbers, there will be more races. Not only are the isolates of the same race not identical, each isolate within itself may show variance.

P. oryzae co-evolved with rice for a very long time. It must be extremely diverse to adapt to the wide genetic base present in rice. The results of the inoculation experiments mentioned above suggest the complex nature of the pathogenicity of the fungus. Studying single-spore progenies from a common origin would be an easy and efficient way to determine diversity. When we inoculated about 50 single-spore cultures, each from two lesions, onto a set of 12 Philippine differentials, we found 14 different reaction patterns (races) from one lesion and eight races from the second lesion. In a further step, we inoculated cultures of 25 daughter conidia from each of two monoconidial cultures. Again each family showed nine and ten different pathogenic patterns. Even conidia produced from hyphal-tip cultures of single cells of one conidium differed in pathogenic patterns. This new dimension of variability was a great surprise to us and more so to other people. Some did not believe or did not wish to believe it was possible, since it rendered all previous race studies almost meaningless. A year later an independent study confirmed this extreme variability. In fact, such variability can be found in several earlier reports, but its significance was not recognized or emphasized. It seems that the physiologic races of *P. oryzae* are temporary or transitory phenomena. We appreciate the words of W. B. Hewitt: "If we merely follow the beaten path or just sit in our canoe and float with the stream relying on the imagination of

others . . . and/or doing more of what has been done, then life is but an empty dream" (3).

THE HORIZONTAL RESISTANCE BANDWAGON

Plant pathology, like other professions, has its changing fashions. Horizontal resistance (HR) has been popular in the past 20 years. Many rice pathologists have been trying to find horizontal resistance to rice blast. Others have offered hypotheses on how it should be done. Most of their theories sound good on paper but are difficult to achieve in practice. Some plant pathologists have had strong opinions on studying horizontal resistance that discredited conventional resistance study. Unfortunately, despite these efforts, we have found no practical way to measure horizontal resistance to rice blast. Some believed that measuring the rate of disease development was a sure method. By measuring the rate of development, several rice cultivars were considered to have good horizontal resistance in West Africa. At least two of them, Blue Bonnet and Tainan 8, were well-known susceptible cultivars in their respective homelands. The races of *P. oryzae* have patterns of geographical distribution, and in the absence of major pathogenic races the susceptibility of the cultivars could not be shown.

Yorinori & Thurston attempted to find out if certain characteristics that had been shown to be associated with horizontal resistance to late blight in potatoes existed in rice blast (7). They reported that one of the most difficult problems encountered in studying rice blast was the diversity of lesion types observed on the varieties and selections used. Even using a single isolate, wide variation in lesion types was observed on the same variety in both detached leaf and greenhouse inoculations. Reactions varied from highly resistant to highly susceptible. The authors came to no conclusions about what might represent horizontal resistance to *P. oryzae* in rice. Their study confirmed the extreme variability of the organism and illustrated the difference between theory and practice. Finally, many of the so-called field resistant cultivars selected in Japan were very susceptible in the Philippines (6). As yet the answer to the question of horizontal resistance remains illusive.

In our studies on resistance to rice blast, we found something like vertical and horizontal resistance but we described them as qualitative and quantitative. Qualitative indicates the reaction of each rice cultivar to fungus isolates, resistant or susceptible. Quantitiative is the total amount of disease, i.e. the number of lesions produced. Qualitative resembles vertical, and quantitative horizontal, reactions.

In one of our studies, isolates were inoculated on the cultivars from which they were isolated. The results showed that on the cultivars that had a broad spectrum of resistance, i.e. resistant to most isolates, small numbers of lesions developed. On the other hand, cultivars with narrow spectrums of resistance

developed large numbers of lesions. For instance, when 37 isolates from Tetep, a cultivar of broad-spectrum resistance, were inoculated back onto Tetep, five produced no lesions, 17 produced less than one lesion (average) per plant, others produced a few lesions, and two produced 14 and 16 lesions. The average was two lesions per plant. In the same experiment, a susceptible cultivar, Khao-teh-haeng 17, used as a control, produced 10–67 lesions on each seedling, with an average of 33. Similar studies with other rice cultivars disclosed that the resistance spectrum and the number of lesions were always negatively correlated.

We further found that cultivars with broad spectrums of resistance as identified in the international blast nurseries (i.e. resistant in most test locations) had fewer lesions, while those with narrow spectrums of resistance had the reverse. Regardless of whether the isolates were from the field or from single-spore cultures or whether the amount of disease was measured in the field or in artificial inoculations, there was always a close negative correlation between the spectrum of resistance and the number of lesions.

Some 240 pathogenic races have been identified in the Philippines, and we know exactly the number of races to which each differential cultivar is resistant. The spectrum of resistance of the differential cultivar varies from 10–90%. Whether they were inoculated artificially or naturally infected, the amount of disease was again negatively correlated with the spectrum of resistance.

From these many results, we assumed that cultivars resistant to most isolates (races) have more genes for resistance, and they appear to have horizontal resistance. This seems to fit very well with R. R. Nelson's hypothesis that the accumulation of vertical resistance may confer horizontal resistance. The reactions basically are vertical, but the end results appear to be horizontal. In other words, if you see only the end results, resistance is horizontal (amount of disease), but when the resistance is analyzed isolate by isolate it is vertical or racial. We prefer to call this stable resistance rather than horizontal resistance. Horizontal resistance in this case seems to be another artifact (2).

THE INTERNATIONAL APPROACH

P. oryzae is capable of producing many pathogenic races. However, the survival and prevalence of a race depends on the availability of a host cultivar. After their long association, *japonica* rice cultivars are readily infected by the prevailing races in Japan, while many *indica* cultivars are resistant. The reverse is true in the tropics. Pathogenic races differ from locality to locality and also from season to season. One of our studies showed that the composition and frequency of races in the IRRI blast nursery differed from month to month, although there were one or two prevailing most of the time. Tests conducted in one country or only a few times cannot assure us of the resistance levels of any

cultivar. Obviously, the best test of resistance is to expose rice cultivars to all existing races at different regions over a period of years through international cooperatives tests.

After a symposium on rice blast disease in 1963, the IRRI assumed the responsibility of coordinating the international blast nurseries (IBN) program, from multiplying the seeds to compilation of the data. Some 30 countries and some 50 or 60 testing stations from all the rice-growing regions of the world have participated. Each year several hundred cultivars are tested. These experiments are the most extensive and efficient way available to test blast resistance, and they are now in their twentieth year. One of the most significant results is the identification of rice cultivars with broad-spectrum resistance. The test cultivars have shown a wide range of resistant reactions, from 20–98% of the tests. Some rice cultivars, such as Tetep and Carreon, have been resistant in 95% or more of the tests and have been consistent year after year.

Many of the cultivars with broad spectrums of resistance were crossed singly or in combination with cultivar IR8, which has a desirable plant type. In some cases nine or ten back crosses with IR8, as well as the progenies in different generations, were screened continuously for many years at the IRRI blast nurseries. The best 50 lines were put into the IBN test in the late 1970s. In the test results over the last five or six years, many lines showed a level of resistance as high as the parents, such as Tetep, Carreon, and *Oryza nivara*. These include lines of IR1416, IR1905, IR3259, IR4227, IR4547, IR5533, IR13429, and others. Some, such as IR4547, are also resistant to other diseases and to some insects. Now we have lines with both high levels of resistance and good plant type. These should be better donors for breeding blast resistance. This exercise also suggests that rice cultivars with a high level of stable resistance can be developed. At present, this can be done most effectively by exposing the selections of one country to the *P. oryzae* strain of a number of cooperating countries over a period of years, as is being done in the IBN.

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